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The Potential for Pulmonary Heat Injury Resulting From the Activation of a Cabin Water Spray System to Fight Aircraft Cabin Fires

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16. Abstract

A cabin water spray system (CWSS) has been suggested as a means of attenuating the severity of smoke and fire commonly associated with aircraft accidents. All aspects of passenger and cabin safety must be considered when evaluating a new safety system or concept. The purposes of this report are to briefly review the pathophysiological changes occurring in the respiratory system as a result of thermal injury and to quantitatively estimate the risk of creating a more hazardous cabin environment by activation of CWSS. Changes in the heat content of the cabin atmosphere resulting from CWSS activation were calculated using parameters consistent with current aircraft and proposals for CWSS design. The results suggest that only a very small volume of the aircraft cabin would have an increase in heat content that could result in thermal injury.

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THE POTENTIAL FOR PULMONARY HEAT INJURY RESULTING FROM THE ACTIVATION OF A CABIN WATER SPRAY SYSTEM TO FIGHT AIRCRAFT CABIN FIRES

I. INTRODUCTION

Fire represents one of the most catastrophic events to which an organism can be subjected. Injury and death from fire can occur due to the incineration of tissue and also by the incapacitating byproducts of the fire environment. The odds of fire injury are increased when individuals are trapped within an enclosed space, such as an aircraft cabin. Testimony to this problem aboard aircraft is the Manchester incident of 1982 in which 52 people lost their lives due to inhalation injury. Efforts continue to develop safety systems which would minimize passenger deaths and injury resulting from aircraft fires. To date, no acceptable solution has been obtained. One suggestion has been to install some type of cabin water spray system (CWSS) in passenger aircraft which can slow the spread of fire, thereby allowing a greater amount of time for passenger evacuation. Due to the cost and complexity of such systems, implementation must be carefully evaluated.

One consideration of CWSS development is its impact on the fire environment. The use of a CWSS might result in the production of steam. Steam may have untoward effects on individuals in the area in which the CWSS was activated, thereby preventing any net benefits on passenger survival. The primary purposes of this paper are to examine the likelihood of 1) the water released from a CWSS being vaporized to some extent and causing injury to the respiratory tract, and 2) the impact of temperature changes in the environment due to such vaporization increasing the risk of thermal injury. Some of this analysis will be based on experimental and clinical studies dealing with thermal injury and some of it on theoretical calculations of heat content within the cabin environment in which a CWSS is heat activated.

II. MECHANISMS OF PULMONARY INJURY DUE TO FIRE

Clinically, there are three categories of burn injury: (1) smoke inhalation, (2) cutaneous burn without smoke inhalation and (3) a combination of 1 & 2 (19). Pulmonary damage due to smoke inhalation and carbon monoxide intoxication is thought to be the leading cause of death in fire victims. Of 12,000 annual deaths in the United States due to fire, 50 to 60 per cent are secondary to inhalation injury (29). Careful study of smoke inhalation and the recognition of its existence did not begin until after the Coconut Grove night club fire which occurred in Massachusetts in 1942. Thirty-six of the 39 individuals that survived transport to the hospital eventually died of pulmonary injury rather than cutaneous burns (2). Concern over this and other fire related catastrophes developed into a significant interest in burn injury during the early 1940's. Investigation of the mechanisms of pulmonary injury, pathophysiological characteristics of the thermally injured respiratory tract, and optimization of diagnostic and treatment protocols has received worldwide investigative attention in the ensuing decades.

A fire represents a variety of chemical reactions, each of which contributes different groups of potentially injurious substances to the breathable atmosphere (28). The nature and amount of these combustion products are determined by the material being burned, the amount of oxygen that is available at different times during the conflagration, and the temperatures attained (18). Because of the fire environment's great complexity (25) it is difficult to make a definitive quantitative assessment of those components of the fire atmosphere most responsible for inhalation injury. Direct injury of the respiratory system may be produced by both the heat content and

Factor	Unfavorable	Favorable
Place burn occurs	Enclosed (room, building, automobile)	Open
Anatomical location of burns	"Respiratory area" involved (mouth, pharynx, nose, nasal hairs)	"Respiratory area" spared
Type of burn	Flame	Liquid, chemical, irradiation
Heavy smoke; products of incomplete combustion	Present	Absent
Steam; high humidity	Present	Absent

Table 1. Factors influencing the incidence and severity of respiratory burns (23).

the chemical characteristics of the gases and particulate matter inhaled. Inhalation injury resulting from fire is found most often with a combination of three conditions: (1) a closed-space accident, (2) the presence of heavy smoke, and (3) a history of unconsciousness (20). Table 1 lists favorable and unfavorable conditions influencing the development of inhalation injury. Note that many of the factors that favor the development of the injury could be expected to be present during an aircraft cabin fire.

Victims of conflagrations frequently sustain more severe life threatening pulmonary injuries than the burns received on the surface of the body. Wound sepsis was once the primary cause of death in burn patients. Since wound sepsis can now be controlled, respiratory complications of inhalation injury and pulmonary sepsis are currently a major cause of death in patients with body surface burns (11, 15). The incidence of significant pulmonary injury has been estimated to be between 15 and 22 percent in burn patient populations (1, 26) with inhalation injuries occurring in approximately one-third of all major burns (11).

Upper airway and lung inhalation injuries are usually secondary to inhalation of gaseous or particulate products of incomplete combustion and rarely are due to heat unless steam has been inhaled. In some instances, respiratory tract damage is confined to the upper air passages with little or no damage to the lungs. It should also be noted that death from inhalation injury can occur without thermal wounds. However, respiratory tract damage is often more severe in burned than in nonburned patients for a given degree of smoke inhalation. Death from inhalation injury is particularly common during the early postburn period (9, 19). The role that thermal, gaseous and particulate matter may play in the development of respiratory damage from fire exposure is presented below.

Thermal Injury

The definitive experimental study addressing the question of mechanism of thermal injury to the respiratory tract during a fire was published in 1945 (18). In this experiment, dogs inhaled hot air (350°C and 500°C), flame from a burner, and live steam. Only the steam resulted in thermal injury at the level of the lung parenchyma. The difference between the hot air and steam treatments were attributed to the low heat-carrying capacity of air coupled with the efficient heat absorption of the upper airway. Based on these observations, the true thermal injury to the lower respiratory tract should be relatively rare. This is substantiated clinically by the observation that inhalation injury below the level of the vocal cords is a relatively uncommon consequence of thermal trauma, occurring in <5% of all burn patients (6, 22, 24). However, thermal injury to the respiratory tract is important.

The unique problems of respiratory tract damage resulting from thermal injury have been recognized throughout the world (14, 27, 30). It has been suggested that classification of pulmonary burns be based on etiology and severity of injury (4) instead of the time of clinical onset of respiratory distress, anatomy, or burn etiology.

Particulate Injury

Except for the rare instance of steam inhalation, direct thermal burns to the respiratory tract below the level of the larynx do not occur in patients (5, 6, 22, 24). The clinical "respiratory burn" is actually a chemical burn, induced by inhalation of suspended particles (smoke) and the toxic products of incomplete combustion (3, 8). This type of injury is a major complication of fire accidents. Chemical burn of the airways occurs upon inhaling the incomplete products of combustion causing surface damage to the larynx, proximal and distal airways and the lung parenchyma (13). The noxious constituents of smoke are believed to stimulate irritant receptors producing bronchoconstriction and to cause chemical injury to the airway mucosa and the alveolar-capillary membrane, thereby producing pulmonary edema. The interaction and impact of gases and other fire byproducts upon physiological function is extensively reviewed elsewhere (16, 25). The probability of respiratory tract injury resulting from inhalation of noxious gases and chemicals is much greater than thermal damage occurring from exposure either to hot air saturated with water vapor or live steam produced by the vaporization of droplets, which could result from use of a CWSS. Some of the morphological changes that could be expected from this type injury are presented below.

III. PATHOPHYSIOLOGICAL RESPONSE TO THERMAL INJURY

Experimentally, evaluation of thermal inhalation injury in dogs has shown that inhalation of hot air (350 and 500°C) causes a thermal tracheitis of the upper trachea without injury to the lower trachea. Inhalation of steam causes thermal injury extending from the trachea to the lung parenchyma (18). Several breaths of steam delivered into the pharynx at a temperature of 100°C causes such a severe local edema within a few hours that the animals die of obstructive asphyxia. Steam produces an immediate pulmonary edema in isolated lung lobes and the fluid transudation reaches its peak within half-an-hour after the inhalation (31). The ulcerations and rapid onset of edema formation observed in these studies is affirmed by clinical observations of inhalation injury.

Inhalation injury has been anatomically divided into three levels: 1) upper airway injury (burn damage limited to the larynx and vocal cords); 2) major airway injury (burn damage involving the tracheobronchial tree) and 3) parenchymal injury (burn damage involving the terminal bronchi and alveolar space) (21). Although the mucous membrane of the entire respiratory tract is potentially susceptible to damage, the infraglottic airway is often spared or injured to a lesser degree than the supraglottic airway because the vocal cords represent an anatomic barrier to the passage of heat into the trachea (13). This factor contributes to the relative rarity of true heat inhalation injury.

Functional and anatomical changes resulting from thermal injury to the respiratory tract have been well documented (7, 10). Heat energy produces immediate injury to the mucosa of the airway and pulmonary parenchyma. There is a rapid loss of fluid volume from the vascular space and concomitant expansion of the interstitial space (edema) immediately following injury. The mucosa develops severe edema, hemorrhage, and ulceration soon after exposure to hot steam. The parenchyma exhibits marginal emphysema and congestion, leading to acute pulmonary edema. Edema formation and congestion occurs more rapidly in steam inhalation injury than in inhalation injuries produced by chemical agents (26). The tracheal lumen accumulates epithelial cells, white blood cells, and mucus, followed by the development of a purulent and necrotizing broncho-pneumonia within 3 to 4 days.

Three major phases of respiratory injury induced by steam burns have been defined (21). The first phase (within the first hour) is identified by coagulation necrosis and an early reactive stage with edema in the tracheobronchial tree and early pulmonary parenchymal edema. During the second phase (up to 24 hours) there is a second reactive stage with development of interstitial and perivascular edema, further sloughing

of mucosa, atelectasis, and hemorrhagic consolidation. Laryngeal edema and laryngospasm leading to extrinsic obstruction of the swollen tissues around the vocal cords often occurs within the first 24 hours (11). The third, or infection, phase (> 24 hours), is indicated by the development of bronchopneumonia behind respiratory tract obstruction secondary to mechanical or functional block. Necrotizing tracheobronchitis also occurs in this phase.

IV. INJURY AND INCAPACITATION DUE TO ENHANCED HEAT CONTENT OF THE ENVIRONMENT

In assessing the toxicity of combustion products in fires, Purser (6) lists heat stroke, body surface burns, and respiratory tract burns as ways in which heat may lead to incapacitation and death in fire victims. The mechanisms and results of respiratory tract burns are discussed in Sections II and III above. With regard to CWSS activation, it is assumed that most individuals are evacuating and/or removed from areas in which the flames could result in direct injury. Therefore, potential body surface or respiratory tract burns would have to result from increased heat content of the environment. Based upon both clinical and experimental research observations, the only way in which heat content could have a significant impact is through the spray of the CWSS being converted to steam or the heated air of the fire environment becoming saturated with this spray. What is the threat of thermal injury resulting from this scenario?

Each incident in which a CWSS is heat activated may be characteristically different. Prediction of potential water spray transformations is difficult. However, within the context of thermal injury, some basic assumptions must be made. Full scale fire testing indicates that the presence of a CWSS lowers the rate of temperature increase under a variety of spray discharge configurations (17). Without water spray, cabin temperature underwent a drastic rise starting at approximately 90 seconds with temperature values of approximately 275°F being obtained at test termination time of five minutes. The current hypothesis holds that optimal CWSS performance requires the use of a zoned system in which water is discharged when air temperatures reach 300°F in a given zone (31). Based on this information, a temperature level of 300°F seems reasonable for use in calculations. For the entire activation and discharge period, a system using between eight and 20 gallons of water is under consideration. Since one goal to be engineered into the final design of the CWSS is to minimize the volume of water necessary for effective operation, a volume of ten gallons will be used for estimation. Cabin volume is estimated to be 7500 ft^3 (Figure 1).

The worst case scenario is that the entire ten gallons of water would be immediately converted to steam while an individual was located within the area of the steam cloud. If the initial temperature of the water in the CWSS was 75°F and the entire 10 gallons in the system were converted to steam at 120°F, ~24,000 kcal would be required. If this heat were supplied by dry air at 300°F, ~10,000 ft^3 of air would be required.

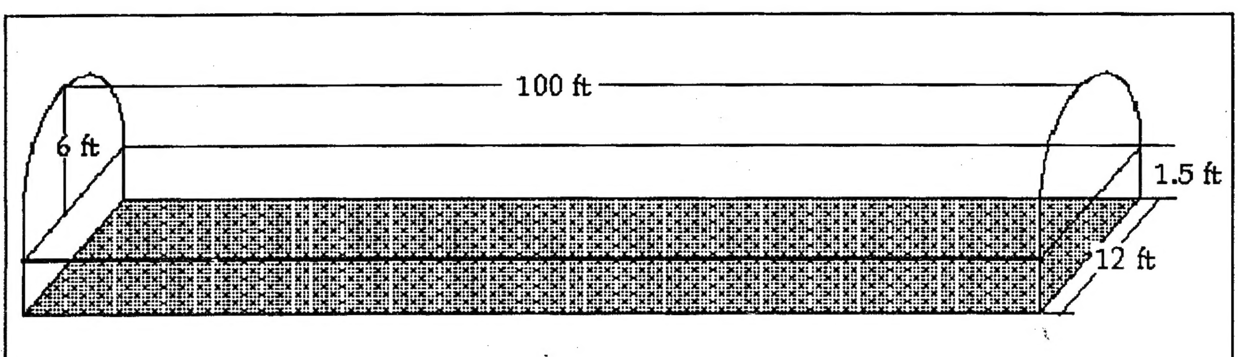

Figure 1. Dimensions used for calculating cabin volume. A bisected cylinder and rectangular shapes were used for volume estimates.

Temperature (°F)	Temperature (°C)	kcal/min	Watts
122	50	1.253	87.42
140	60	1.583	110.49
158	70	2.091	145.94
176	80	2.973	207.44
194	90	4.514	315.09

Table 2. Total heat delivery to the lungs from breathing water vapor saturated air at various temperatures.

However, based on cabin dimensions given above, only 75 ft^3 of air would be present at the hot-air/water-spray interface. This volume of air contains enough heat to convert ~0.076 gallons of water to steam which represents a volume of 0.84 ft^3. Therefore, it appears that the immediate formation of a significant steam cloud is unlikely.

The more likely occurrence would be that as the water is discharged from the system, it saturates the heated air and this hot air and water vapor mixture diffuses "downstream" of the fire. This mixture presents a considerable threat of thermal injury due to the high heat content of water vapor and the potential condensation of this vapor in the lung. Table 2 lists the total heat content of water saturated air in kcal/min and watts based on a ventilatory rate of 10 l/min.

The heat delivered to the lung from inhaling dry or moist air of any temperature is found by subtracting the heat present in the ventilation volume from the heat contained in the air mixture of interest. From the inhaled air mixture temperature, the mean specific heat for both the water and air components can be determined. The density and partial pressure of each component must also be known. From these values the heat per unit volume for each component in the mixture can be calculated. If a saturated water vapor mixture were inhaled at a temperature above 98.6°F, the heat of condensation of the water saturated air must be considered. The heat of condensation represents the majority of heat delivered by a water saturated air mixture.

Figure 2 illustrates the quantity of heat that would be delivered to the lungs based on a ventilatory rate of 10 l/min. As can be seen from the graph, an increasingly larger percentage of heat comes from the condensation of water vapor within the lung as the temperature increases. Experiments studying the impact of inhalation of hot water vapor saturated air have not been done. The expected response to this type of exposure would be reflex closure of the glottis which is the typical response to hot air (13). Therefore, significant damage in the areas of gas exchange would probably not occur. Of course, damage to the skin, nasal passages and airways above the level of the glottis may occur. The extent to which this happens would be dictated by the combination of temperature and exposure time. Potentially, exposure time would be correlated with the volume of saturated air present. This is particularly true if individuals are delayed in evacuating the aircraft cabin. What is the volume that poses this threat if 10 gallons of water are used to produce saturated air at the temperatures listed in Table 2?

Figure 3 presents an analysis for 10 gallons of water being used to create a saturated volume of air at the temperatures listed in Table 2. It is assumed that the saturated air mixture takes the form of a bisected cone as it moves away from the spray area along the ceiling of the aircraft cabin. As one would expect, the lower temperature saturated air is less of a threat. This is due to: 1) a lower heat capacity, and 2) a reduced volume due to a lower density, as can be seen in Figures 2 and

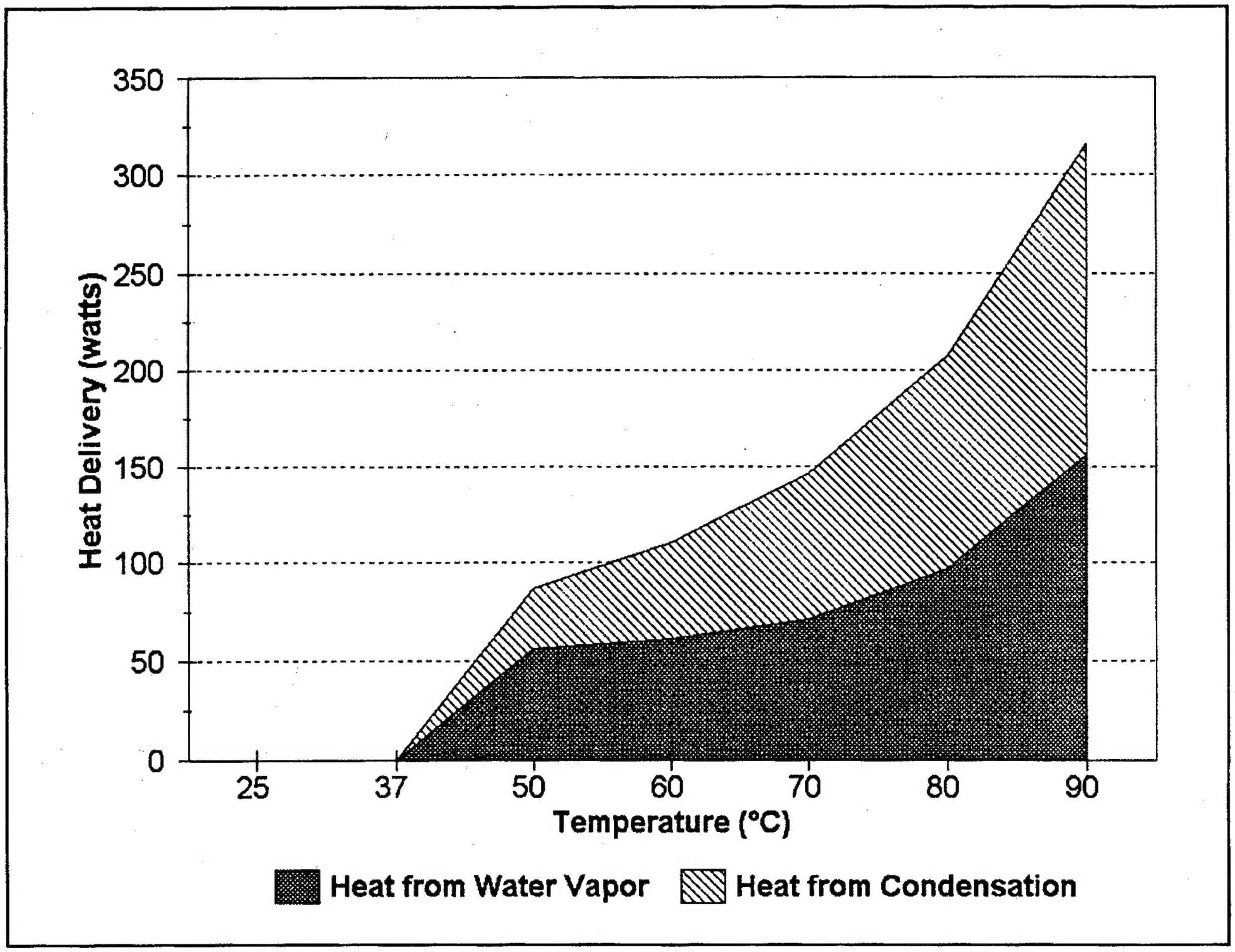

Figure 2. Heat delivery to the lungs based on inhalation of air saturated water vapor.

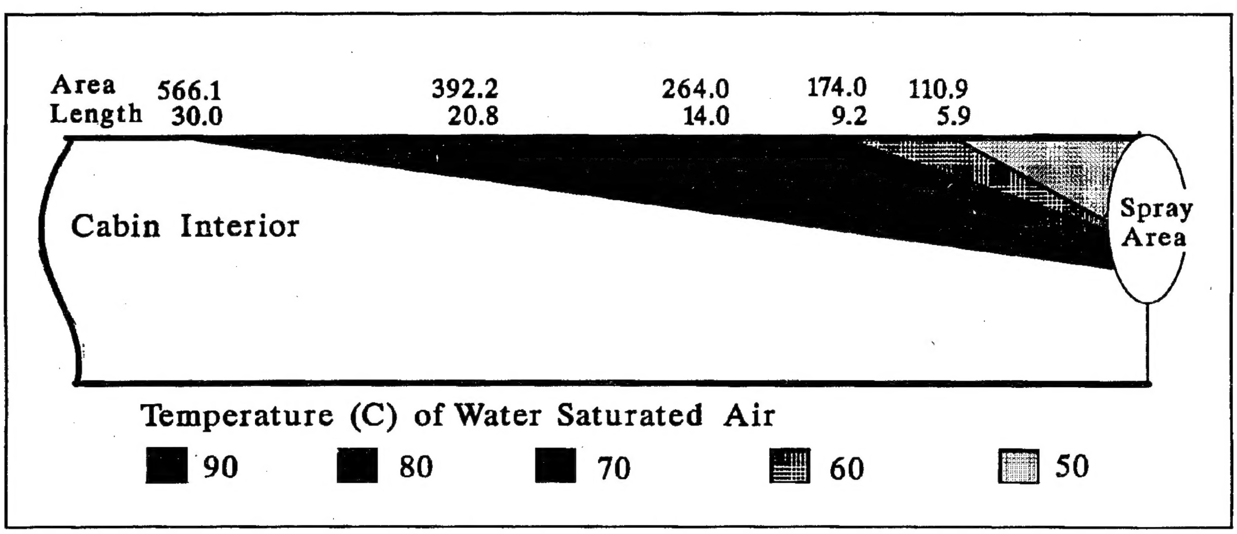

Figure 3. Theoretical distribution of water saturated air in the aircraft cabin.

3, respectively. As temperatures increase, the threat of thermal injury becomes more pronounced. If someone were trapped in a saturated air environment at above 70°C for more than a few seconds, it is very likely that serious injury or death would occur. However, it should be noted that the volume of the highest temperature saturated air represents only 7.6% of the total cabin volume. Furthermore, if the assumption that the high heat area is distributed along the top of the cabin as depicted in Figure 3, much of the high heat exposure could be avoided by crouching or crawling near the floor during evacuation.

CONCLUSIONS

Based on the worst case analysis presented above, the risk due to increased latent heat in the environment resulting from activation of a CWSS is relatively small. Although a potential hazard from steam and hot water vapor saturated air does exist, exposure to these conditions for more than a second or two is highly unlikely and could theoretically be avoided by maintaining the correct posture and quickly evacuating the aircraft. It should be noted that if the CWSS were not present, the heat content of the environment would be significantly higher. The water of the spray is absorbing heat which is, in essence, what produces a significantly lower rate of temperature rise in the cabin when a CWSS is activated (30). The fact that a relatively small, potentially hazardous thermal environment may be produced seems inconsequential in comparison to an uncontrolled fire environment.

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